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## Water Intoxication in a Beer Drinker

BEER PRODUCES A NUMBER of well known physiologic effects, including a water diuresis which is due to an inhibition of anti-diuretic hormone (ADH) release by both the alcohol<sup>1</sup> and the water contained in beer, and a central nervous system intoxication which is ordinarily due only to the alcohol. Elsewhere in this issue of CALIFORNIA MEDICINE, Gwinup et al report the (fortunately) uncommon association of beer ingestion and water intoxication.

A 46-year-old man with a history of having drunk 3 to 6 liters of beer daily for many years presented to the hospital with mental confusion on three separate occasions over a three-year period, and on each occasion he was found to have significant hyponatremia. Serum osmolality was low, demonstrating dilution of total extracellular solutes by water, and urine osmolality was quite high, indicating inappropriate ADH release; however, preliminary study failed to reveal any of the recognized causes of inappropriate ADH release.<sup>2</sup> If it is assumed that the patient ordinarily tolerated his large daily beer intake without suffering water intoxication, the most likely diagnosis is intermittent or temporary inappropriate ADH release of uncertain cause.

An attempt was made, in the present case, to assess the role played by the ingestion of 5 liters of beer a day, by comparing the effects of one week of beer ingestion, one week of an equivalent amount of water ingestion, and one week of concentrated alcohol ingestion (about 220 grams a day, the amount present in 5 liters of 4.6 vol% beer). Beer ingestion was accompanied by a rising urine osmolality as well as

progressive weight gain and hyponatremia which abated abruptly when beer was discontinued. Water ingestion was accompanied by a very slowly falling urine osmolality, as well as some water retention and hyponatremia which abated as the urine gradually became very slightly hypotonic during the last few days of water ingestion. Alcohol ingestion apparently failed to produce either a hypotonic urine or a detectable change in water balance. The authors concluded that beer, per se, may have produced the inappropriately concentrated urine, but allowed for the possibility that the different effects observed between beer ingestion and water ingestion may not have been related to any such effects of beer independently of its water content. What makes the latter possibility attractive is that an abnormality of ADH release, not dependent on beer intake, is strongly suggested by the apparent failure of water or alcohol to result in the production of maximally dilute urine. Furthermore, as was mentioned, the patient may well have tolerated large beer loads on many occasions outside the hospital without developing water intoxication. For these reasons, it seems not unlikely that a non-osmotic factor other than beer—for example, acute anxiety, unusual excitement, abrupt increase in tobacco use, and certain drugs<sup>2</sup>—is causing intermittent and variable release of ADH which resulted in the patterns observed during the periods of study, and which, out of hospital, resulted in water intoxication whenever sufficiently large amounts of water, in the form of beer, also happened to have been ingested. If this interpretation is correct, the role played by beer in the present case is well summarized by the motto appearing on the label of the test beer employed by the authors—"It's the Water."

Finally, although the association of excessive beer intake and water intoxication is certainly an oddity, the present case should serve as a reminder of an entity which, in all likelihood, is not an oddity—that is, temporary inappropriate ADH release, excessive "free-water" intake, and water intoxication.

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